TABLE 1. Pressure Ratio of Cell Suspension to Buffer After 6 Minutes' Filtration

<table>
<thead>
<tr>
<th>Cells</th>
<th>Controls (n=10)</th>
<th>Cerebral infarction patients (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Granulocytes</td>
<td>4.73±0.41</td>
<td>5.60±0.72*</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>5.74±1.18</td>
<td>6.28±0.76</td>
</tr>
<tr>
<td>Monocytes</td>
<td>11.46±2.02</td>
<td>15.98±1.83*</td>
</tr>
</tbody>
</table>

Data are mean±SD. *p<0.01 different from control.

cyte and granulocyte subpopulations, requires continued study to understand further their role in acute cerebral ischemia.

References


To Shunt or Not to Shunt: The Controversy Continues

To the Editor:

We read with interest the report of Drs. Gumerlock and Neuwelt entitled “Carotid endarterectomy: To shunt or not to shunt,” which appeared to indicate that routine bypass shunting during carotid endarterectomy significantly reduces the risk of intraoperative neurologic deficit.1 We recently completed a study of 103 patients undergoing carotid endarterectomy, who were monitored with a two-channel computerized electroencephalographic/compressed spectral array analysis. As best we can tell from the letter, their results are in keeping with the published literature. Since it remains impossible to determine accurately when to use a shunt based on variable monitoring criteria, the only currently assessable option is in fact the decision on whether to shunt. As we have shown in our randomized prospective study using this decision as the only criterion, there are significant benefits to the use of a shunt. We certainly agree with Drs. Tempelhoff and Modica that their results are difficult to interpret and that the answer to the criteria question remains elusive.

Correction: Dr. Kim Wayson’s name did not appear in our resident acknowledgements,1 and we would like to recognize his invaluable help with the study.

Edward A. Neuwelt, MD
Division of Neurosurgery
Department of Biochemistry
University of Missouri–Columbia
Columbia, Missouri

References


Role of Dopamine in Ischemic Neuronal Damage

To the Editor:

Kawano et al. have recently described a large increase in extracellular dopamine release during ischemia in the stratum of spontaneously hypertensive stroke-resistant rats. The authors hypothesized that the excessive leakage of dopamine may be a causal factor in the development of postischemic neuronal damage. The possibility that dopaminergic neurotransmission might contribute to the vulnerability of the stratum during ischemia has been clearly established by some of our recent sequential studies. Using the microdialysis technique in rats subjected to 20 minutes of four-vessel occlusion, we documented an acute and massive increase of dopamine release into the striatal extracellular space.2 Dopamine deafferentation, by prior unilateral sub-
To shunt or not to shunt: the controversy continues.
R Tempelhoff and P A Modica

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